Carvacrol Induces Apoptosis via Caspase Activation in HL-60 human leukemia cells.

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ABSTRACT

Background

One of the central mechanisms through which cancer progresses is by favorably regulating cell proliferation over apoptosis. The cancer cells evade apoptosis by preventing the activation of caspase-3 dependent cell death. However, many plant-derived secondary metabolites have demonstrated their anti-cancer effect. In regard, carvacrol, a phenolic monoterpenoid, is present in essential oils of plants such as oregano (Origanum vulgare), thyme (Thymus vulgaris), and wild bergamot (Citrus aurantium bergamia) and has been reported to have a wide range of therapeutic uses like anti-bacterial, anti-fungal, insecticidal, and antioxidant activities. Hence, we sought to study the molecular mechanism of carvacrol induced apoptosis in human leukemia HL-60 cells.

Methodology and Results

MTT assay was performed to analyze the cytotoxic effect of carvacrol and was found to be toxic in the range of 20-100mM/ml. The main cytotoxic effect appears to be attributable to its induction of apoptotic cell death as assessed by TUNEL assay. Exposure of the cultured cells to carvacrol (100 mM/ml) led to an increase in malonyldialdehyde (MDA) production, an indicator of free radical formation, and reduction in the level of different antioxidant enzymes including reduced glutathione (GSH) and superoxide dismutase (SOD) ($p < 0.05$). Further studies revealed that the dissipation of mitochondrial membrane potential of intact cells is accompanied by the activation of caspase-3. Conclusion: Our findings indicate that carvacrol-induced cellular apoptosis by collapsing mitochondrial membrane potential, generating free radicals, and depleting intracellular antioxidant enzyme levels, thereby indicating its potential for cancer treatment.

Keywords: Carvacol, Phytochemicals, Anti-cancer drug

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